WHAT IS CLAIMED IS:

1. A formulation of a pharmaceutical composition comprising a human recombinant α-L-iduronidase or biologically active fragments or mutein thereof with a purity of greater than 99%, or in combination with a pharmaceutically suitable carrier.

2. The formulation of claim 1, wherein said recombinant α-L-iduronidase or biologically active fragments or mutein thereof with a specific activity of greater than about 240,000 units per mlligram protein.

3. The pharmaceutical composition of claim 1 further comprising a sodium chloride solution, a buffer and polysorbate 80.

4. The pharmaceutical composition of claim 1 wherein said human recombinant α-L-iduronidase or mutein thereof is present at a concentration range of about 80 to 150 units per mL.

5. The pharmaceutical composition of claim 1 wherein said human recombinant α-L-iduronidase or mutein thereof is present at a concentration of about 100 units per mL.

6. The pharmaceutical composition of claim 3 wherein said sodium chloride solution is at a concentration of about 150 mM.

7. The pharmaceutical composition of claim 3 wherein said buffer is a sodium phosphate monobasic buffer at a concentration of about 92 mM.

8. The pharmaceutical composition of claim 3 wherein said buffer is a sodium phosphate dibasic buffer at a concentration of about 8 mM.

9. The pharmaceutical composition of claim 3 after dilution into the dosage form wherein said human albumin is present at a concentration of at least about 1 mg/mL.

10. The pharmaceutical composition of Claim 9 wherein human albumin is used to prevent or reduce acute allergic or complement mediated reactions in said human subject.

11. The pharmaceutical composition of claim 3 wherein the pH of said solution is maintained at about 5.8.

12. The pharmaceutical composition of claim 3 wherein said polysorbate 80 is maintained at 10 μM/mL.

13. The pharmaceutical composition of claim 12 wherein said polysorbate is required to stabilize the protein in the final product.

14. A method of treating diseases caused all or in part by a deficiency in α-L-iduronidase, comprising the steps of:

(a) administering said formulation of Claim 3 to a human subject in need thereof;

(b) optimizing said treatment by assessment of primary efficacy endpoints;

(c) optimizing said treatment by assessment of secondary efficacy endpoints;

(d) optimizing said treatment by assessment of tertiary efficacy endpoints; and

(e) optimizing treatment by assessment of safety endpoints.

15. The method of Claim 14, wherein said primary efficacy endpoints are selected from the group consisting of percent predicted forced vital capacity and six-minute walk distance.

16. The method of Claim 14, wherein said secondary efficacy endpoints are selected from the group consisting of apnea/hypopnea index, liver organ volume, disability score index, and joint range of motion.

17. The method of Claim 14, wherein said tertiary efficacy endpoints are selected from the group consisting of urinary glucosaminoglycan levels, total respiratory event index, pain, joint range of motion, quality of life, growth in prepubertal patients, visual acuity, echocardiogram, and forced expiratory volume.

18. The method of Claim 14 wherein the disease is mucopolysaccharidosis.

19. The method of Claim 14 wherein the disease is MPS I.

20. The method of Claim 14 wherein the disease is selected from the group consisting of: Hurler's disease, Scheie syndrome and Hurler-Scheie syndrome.

21. The method of Claim 14 wherein said subject suffering from the disease demonstrates about 1% or less of a normal α-L-iduronidase activity.

22. The method of Claim 14 wherein a dose of at least about 100 units per kilogram said human recombinant α-L-iduronidase is administered weekly to a patient suffering from a deficiency thereof.

23. The method of Claim 22, wherein said dose is administered over a four-hour infusion.

24. The method of Claim 14 wherein said administering is the slow infusion of at least 0.5 mg/kg of said formulation for about an hour, followed by a rapid two-hour infusion rate.

25. The method of Claim 24 wherein said infusion is used to minimize complement mediation clinical allergic reactions.

26. The method of Claim 14 wherein said treatment with human recombinant α-L-iduronidase reduces lysosomal storage.

27. The method of Claim 14 wherein said treatment causes improvement in said endpoints of said human subjects.

28. The method of Claim 14 wherein said treatment results in improvement in percent forced vital capacity, improvement in six-minute walk, normalization of liver volume and urinary glycosaminoglycan excretion, reduction in spleen size and apnea/hypopnea events, increase in height and growth velocity in prepubertal patients, improvement in shoulder flexion and elbow and knee extension, improvement in symptoms related to cardiac function, and improvement in endurance and limitations of daily activities.